

# Trypanosoma cruzi calreticulin inhibits the complement lectin pathway activation by direct interaction with L-Ficolin

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Trypanosoma cruzi, the agent of Chagas' disease, the sixth neglected tropical disease worldwide, infects 10-12 million people in Latin America. Differently from T. cruzi epimastigotes, trypomastigotes are complement-resistant and infective. CRPs, T-DAF, sialic acid and lipases explain at least part of this resistance. In vitro, T. cruzi calreticulin (TcCRT), a chaperone molecule that translocates from the ER to the parasite surface: (a) Inhibits the human classical complement activation, by interacting with C1, (b) As a consequence, an increase in infectivity is evident and, (c) It inhibits angiogenesis and tumor growth. We report here that TcCRT also binds to the L-Ficolin collagenous portion, thus inhibiting approximately between 35 and 64% of the human complement lectin pathway activation, initiated by L-Ficolin, a property not shared by H-Ficolin. While L-Ficolin binds to 60% of trypomastigotes and to 24% of epimastigotes, 50% of the former and 4% of the latter display TcCRT on the